ZINC PHOSPHIDE/PHOSPHINE
TECHNICAL FACT SHEET

NPIC Technical Fact Sheets provide information that is complex and intended for individuals with a scientific background and/or familiarity with toxicology and risk assessment. This document is intended to promote informed decision-making. Please refer to the General Fact Sheet for less technical information.

Chemical Class and Type:
- Zinc phosphide is an inorganic compound that is used in pesticide products as a rodenticide. The International Union of Pure and Applied Chemistry (IUPAC) chemical name is trizinc diphosphide, and the Chemical Abstracts Service (CAS) registry number is 1314-84-7.
- Zinc phosphide was first registered for use as a pesticide in the United States by the U.S. Department of Agriculture (USDA) in 1947. Subsequently, a Registration Standard for zinc phosphide was issued by the United States Environmental Protection Agency (U.S. EPA) in 1982. A Reregistration Eligibility Decision, or RED, was issued by the U.S. EPA in 1998. See the text box on Laboratory Testing.
- Zinc phosphide converts to phosphine gas in the presence of moisture and acid in the stomach. The toxicity of zinc phosphide is due to phosphine gas exposure. This fact sheet will discuss both zinc phosphide and phosphine.
- Phospine in this fact sheet refers to the gaseous form of this compound. The chemical reaction that releases phosphine is: $\text{Zn}_3\text{P}_2 + 6\text{H} \rightarrow \text{PH}_3 + 3\text{Zn}^{2+}$
- Aluminum phosphide and magnesium phosphide also react with water to produce phosphine. These compounds are frequently used to fumigate grain storage facilities. They may be used as rodenticides as well as insecticides.
- Phosphine is used in the electronics industry and in the manufacture of organophosphate insecticides. Phosphine may also be produced during the manufacture of methamphetamine depending on the method used.
- Under natural conditions, phosphine can be produced during the anaerobic decomposition of organic matter, such as in the production of swamp gas. It is also produced in sewage treatment plant sediments.

Physical / Chemical Properties:
- Zinc phosphide is a gray-black powder with an odor similar to garlic. Phosphine is a colorless and flammable gas. The odor of industrial or technical grade phosphine gas is similar to garlic or rotting fish, but phosphine in its pure form is odorless.
- Humans can begin to detect phosphine at 2 ppm, although toxicity can occur at lower concentrations.
- Vapor pressure: When dry, zinc phosphide has negligible vapor pressure. The vapor pressure of phosphine is $2.93 \times 10^4$ mmHg at 25°C.
- Octanol-Water Partition Coefficient (log $K_{ow}$): Not found. Zinc phosphide is not soluble in alcohol or water.
- Henry’s constant: Zinc phosphide, none found; phosphine, $2.44 \times 10^{-2}$ atm·m$^3$/mol.
- Molecular weight (g/mol): Zinc phosphide, 258.12 g/mol; phosphine, 34 g/mol.
- Solubility (water): Zinc phosphide is practically insoluble in water and insoluble in alcohol. Phosphine is considered slightly soluble in water.
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- Soil Sorption Coefficient ($K_{oc}$): Not found.

Uses:
- Rodenticides containing zinc phosphide are used in both agricultural and residential settings. Formulations include bait pellets, granules, dust, and tracking powders. Uses for individual products containing zinc phosphide vary widely. Always read and follow the label when applying pesticide products.
- Signal words for products containing zinc phosphide may range from Caution to Danger. The signal word reflects the combined toxicity of the active ingredient and other ingredients in the product. See the pesticide label on the product and refer to the NPIC fact sheets on Signal Words and Inert or “Other” Ingredients.
- To find a list of products containing zinc phosphide which are registered in your state, visit the website http://npic.orst.edu/reg/state_agencies.html and search by “active ingredient.”

Mode of Action:
Target Organisms
- Rodenticide baits containing zinc phosphide must be ingested to be effective. They are used to control both commensal rodents such as house mice and rats, and “field” rodents such as voles, ground squirrels, pocket gophers, and prairie dogs. Jack rabbits are also targeted pests.
- The toxicity of zinc phosphide is due to the production of phosphine. This is also true of aluminum phosphide and magnesium phosphide. Zinc phosphide requires acidic conditions for appreciable hydrolysis and subsequent formation of phosphine, whereas aluminum phosphide and magnesium phosphide will hydrolyze to form phosphine in neutral pH.
- Hydrolysis is strongly pH-dependent for zinc phosphide. At pH 4, 7.1% of zinc phosphide hydrolyzed in 12 hours, whereas 38.8% hydrolyzed at pH 2 over the same period.
- Once released in the gastrointestinal tract, phosphine gas is absorbed along with zinc.
- Phosphine disrupts mitochondrial respiration once it is absorbed. The exact mechanism is unknown, but phosphine appears to block protein and enzyme synthesis. Phosphine is a cytochrome C oxidase inhibitor, but this does not appear to be its sole mechanism of toxicity.
- Other possible mechanisms of action for phosphine include creation of hydroxyl radicals while simultaneously inhibiting catalase and peroxidase, corrosion of exposed tissues, and metal toxicity from the zinc, magnesium or aluminum. Anticholinesterase activity by phosphine has also been suggested, but evidence for this has been mixed.

Non-target Organisms
- Ingestion of zinc phosphide by non-target mammals or birds leads to the liberation of phosphine and subsequent toxicity in the same manner that target mammals are affected. Zinc phosphide is highly toxic to sheep, cows, and goats as well as non-ruminants.
- Non-target organisms such as rabbits that are unable to vomit are at particular risk of phosphine poisoning.
- Phosphine production is increased at decreasing pH. Animals that have recently eaten and have food in their stomachs are at greater risk than animals with empty stomachs prior to consuming zinc phosphide. This is because of the secretion of gastric acid into the stomach for digestion.
- Animals with relatively constant release of gastric acid regardless of food intake, such as rats, are at greater risk than animals such as dogs whose gastric acid is released only following ingestion of food.
- Zinc phosphide is not expected to pose a secondary poisoning hazard because of its rapid breakdown in the bodies of animals that ingested it directly. However, experimental oral exposures to animals poisoned by zinc phosphide have led to secondary poisonings in both cats and dogs.
Secondary poisoning appears to be rare in wildlife, although it has occurred in experimental settings. Risk appears to be dependent at least in part on the consumption of the gastrointestinal tract of the poisoned animal by either a predator or scavenger. Domestic dogs and cats appear to be more likely to consume the gastrointestinal tract of poisoned prey than are wild animals.19

### Acute Toxicity:

**Oral**

- The oral LD$_{50}$ for rats was determined to be 21 mg/kg, with a range of 12-35 mg/kg in one study, and 43-56 mg/kg in another.20,21 The U.S. EPA considered zinc phosphide to be highly toxic via oral exposure.2 See the text boxes on Toxicity Classification and LD$_{50}$/LC$_{50}$.

- The oral LD$_{50}$ for sheep is 60-70 mg/kg.16

- Oral LD$_{50}$ values were compiled for wild mammals and ranged from 8 mg/kg for kangaroo rats (Dipodomys spectabilis) and black-tailed jackrabbits (Lepus californicus) to 93 mg/kg for a kit fox (Vulpes macrotus mutica).19

**Dermal**

- The dermal LD$_{50}$ in rabbits was determined to be 2000-5000 mg/kg.22 Zinc phosphide is considered to be low in toxicity-based on these results.2

- Eye irritation tests performed with zinc phosphide on rabbits resulted in discharge, chemosis or swelling of the eyelid and eye surface tissue, and some redness in the conjunctiva. The U.S. EPA considered zinc phosphide to be very low in toxicity for eye irritation.2
Researchers applied zinc phosphide to the skin of rabbits to determine if it is a skin irritant. Zinc phosphide was found to be non-irritating. Based on the low dermal toxicity and lack of dermal irritation, the U.S. EPA waived the skin sensitization tests.

Inhalation
- The U.S. EPA waived the re-registration requirement of determining an inhalation LC₅₀ for zinc phosphide and considered it highly toxic via inhalation exposure.
- Rats exposed to 0, 2.5, 5.0, and 10.0 ppm phosphine for 6 hours all survived, although some animals exhibited nasal discharge during the exposure. Nasal discharge cleared after the exposure ended, and no exposure-related effects were noted 14 days after exposure.

Signs of Toxicity - Animals
- Animals that ingest zinc phosphide may begin showing clinical signs within 1 to 4 hours. Early signs of exposure include loss of appetite and depressed activity followed by vomiting and painful retching. These signs progress to anxiousness, ataxia or uncoordinated movements, weakness, labored breathing, thrashing, muscle tremors and convulsions.
- Onset of signs may be delayed for up to 12 hours or more in animals who consumed the bait without any other food in their stomachs. Gastric acid release in animals that have recently eaten causes more rapid release of phosphine.
- The vomit of poisoned animals may contain blood. The vomit can also include phosphine, which can be dangerous to humans at levels below which its odor can be detected.
- Rats poisoned with 10 mg/kg aluminum phosphide administered intraperitoneally demonstrated a drop of 47% in cholinesterase activity.
- Researchers exposed rats to aluminum phosphide by inserting it through the stomach wall. Treated rats developed methemoglobinemia.

Signs of Toxicity - Humans
- Zinc phosphide dust may release phosphine once it contacts the moist tissues of the respiratory tract if the dust is inhaled, resulting in pulmonary edema and cardiotoxicity. If ingested, zinc phosphide releases phosphine in the gut and may cause headache, dizziness, fatigue, nausea and vomiting, cough, dyspnea, chest tightness, and thirst. Other signs include liver failure, jaundice, loss of ability to urinate, tetany, delirium, convulsions, coma, and death.
- Death in humans from fatal doses may be delayed for 30 hours after exposure, with the majority of tissue damage occurring in the liver, kidneys and heart.
- Victims of lethal phosphine exposure were found to have liver, myocardial, and alveolar cell necrosis, pulmonary edema and microscopic pulmonary congestion, and anoxic damage in the brain.
- Hyperglycemia following exposure to phosphine has also been reported. In a study of 45 patients admitted to the hospital following aluminum phosphide poisoning, researchers noted that increased blood glucose levels or hyperglycemia were associated with fatal outcomes.
- Elevated levels of the enzyme creatine phosphokinase were found in two instances of severe poisoning by phosphine.
- Aluminum and phosphine have been shown to interfere with acetylcholinesterase in humans, but the impact of this inhibition on the toxicity is not clear.
- Always follow label instructions and take steps to avoid exposure. If any exposures occur, be sure to follow the First Aid instructions on the product label carefully. For additional treatment advice, contact the Poison Control Center at 1-800-222-1222. If you wish to discuss an incident with the National Pesticide Information Center, please call 1-800-858-7378.
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Chronic Toxicity:

Animals
• Rats inhaled phosphine 5 days a week, 6 hours a day, for up to 2 years. The concentrations tested were 0.3, 1.0, and 3.0 ppm. The researchers did not detect any toxic or carcinogenic effects from these exposures.30

• Rats inhaled phosphine at concentrations of 0, 0.3, 1.0, and 3.0 ppm for 6 hours a day for 13 weeks. An additional group of rats was exposed to 5.0 ppm of phosphine for 6 hours a day for 13 days. Rats exposed to 1.0 ppm or more gained less weight and consumed less food than controls. Minor changes in blood parameters and kidney function were noted in animals in the 3.0 and 5.0 ppm exposure groups, although all effects disappeared after cessation of exposure.23

• Mice inhaled phosphine at concentrations of 0, 0.3, 1.0, and 4.5 ppm for 6 hours per day, 5 days per week for 13 weeks. Dose-related decrease in weight gain was noted particularly in female mice. Masses of male kidneys, heart, brain, and lungs were less than those of controls at all dose levels, whereas females’ organs increased in mass at the 4.5 ppm exposure level.31

• Researchers fed rats baits containing either 0.02 or 0.03% zinc phosphide for up to 58 or 31 days, respectively. During the first week, rats in the treatment groups gained less weight than control group rats. All rats in the 0.03% treatment group died, and two rats from the lower-dose group died by the end of the study. Researchers found liver injury in rats from the high-dose group. Lung damage was also noted.17

• A 90-day subchronic study was performed on rats. Researchers administered zinc phosphide by gavage (stomach tube) at doses of 0.1, 1.0, or 3.0 mg/kg/day. Animals dosed with 1.0 or 3.0 mg/kg/day showed excess salivation and lowered external body temperature. Hydronephrosis, or swelling of the kidney with excess urine, was noted in the male rats in the 1.0 and 3.0 mg/kg/day dose groups, and males in the highest dose group also developed kidney infections. The NOEL was established at 0.1 mg/kg/day.2 See the text boxes on NOAEL, NOEL, LOAEL, and LOEL.

• The chronic RfD for zinc phosphide was set at 0.0001 mg/kg/day based on the subchronic exposure study in rats.2 See the text box on Reference Dose (RfD) (page 9).

• Rats were dosed with 40, 80, or 160 mg zinc phosphide or fed bait containing 2% zinc phosphide prior to being offered to ferrets. Ferrets were fed a total of five rats over a 10-day period. Ferrets fed control rats readily ate the gastrointestinal tracts, but ferrets fed dosed rats began to avoid eating the gastrointestinal tracts of poisoned rats after 4 days, particularly those in the high-dose group. Blood chemistry of ferrets fed poisoned rats had reduced hemoglobin-to-iron ratios and increased triglyceride concentrations compared to control ferrets, although there was considerable individual variation.32

• Researchers fed gray foxes (Urocyon cinereoargenteus) and red foxes (Vulpes vulpes) exclusively on voles killed with zinc phosphide for 3 days. Dosed foxes ate less food and cached fewer poisoned voles than they had when fed unpoisoned voles prior to the study.33

Humans
• Chronic, low-level inhalation or oral exposures to zinc phosphide in people have been associated with weakness, anemia, toothache, necrosis of the jaw bones and associated swelling, weight loss, and spontaneous fractures.4

• Blood samples taken from fumigant applicators and control subjects were examined for chromosomal abnormalities. Researchers collected samples during the application season, and 6 weeks to 3 months after application. Fumigant applicators who had been exposed to phosphine had more chromosome abnormalities than control subjects during the application season. Chromosomal rearrangements were more common in phosphine applicators than in controls 3 months after exposure.34

• More recent investigations repeated the earlier work above, but found no such differences.35,36 One group of researchers hypothesized that improved personal protective equipment practices were responsible for the change.36 Other researchers reported that chromosomal breaks were more prevalent in men who used fumigants relative to controls, but these applicators also used insecticides and herbicides.37
Endocrine Disruption:

- No data were found on the ability of zinc phosphide or phosphine to disrupt the endocrine system.

Carcinogenicity:

Animals

- The U.S. EPA waived requirements for carcinogenicity studies for zinc phosphide because chronic exposure is not expected.²

- Rats were fed diets for 2 years that had been fumigated with phosphine at rates of 48 and 90 g/metric ton for 48 and 72 hours, respectively. Feed was stored frozen following fumigation and residues at time of thawing averaged 1 ppm. Residues were expected to begin to dissipate at thawing, and were therefore unknown at time of consumption. No signs of carcinogenicity were noted.³⁸

- Rats exposed to phosphine through whole-body inhalation at concentrations of 0.3, 1.0, and 3.0 ppm for up to 2 years exhibited no carcinogenic effects.³⁰

Humans

- The U.S. EPA determined that chronic exposure to zinc phosphide should be negligible and therefore waived carcinogenicity testing requirements for reregistration.² See the text box on Cancer.

- No human data were found on carcinogenic effects of zinc phosphide or phosphine.

Reproductive or Teratogenic Effects:

Animals

- Researchers dosed 25 pregnant female rats per group daily by stomach tube at doses of 1, 2, or 4 mg/kg/day during the second week of the pregnancy. Nine of the rats in the highest dose group died although the cause of death was not determined. Rats in the highest dose group also ate less and lost weight in the first half of the week. Both parameters returned to pre-study levels by the end of the treatment period. The maternal NOEL was established at 2 mg/kg/day.¹⁹

- Researchers exposed 24 pregnant female rats to phosphine for the 20 days of gestation at concentrations of 0, 0.03, 0.33, 2.80, 4.90, and 7.50 ppm in whole-body exposure tests. Fourteen of the females died by day 10 in the high-dose group. No treatment-related effects were noted in the dams of all other exposure groups.²³

- Ten male and 10 female adult rats were fed 0.03% zinc phosphide for 22 days. One male and 4 females died before the end of the exposure. All of the surviving rats maintained their fertility despite the exposure.¹⁷

Humans

- No human data were found on the teratogenic or reproductive effects of zinc phosphide or phosphine exposure.

Fate in the Body:

Absorption

- Absorption of phosphine occurs through inhalation. It also occurs across the gastro-intestinal tract following ingestion of zinc phosphide and subsequent production of phosphine. Dermal absorption of zinc phosphide is low.⁴,¹⁶

- Aluminum phosphide and magnesium phosphide are expected to hydrolyze to phosphine upon contact with moist respiratory membranes, and the phosphine can then be absorbed by the lungs.⁴
• Inhalation exposure to zinc phosphide may lead to exposure through the gastrointestinal tract via particulate clearance mechanisms in the lungs, which could result in ingestion of particulate matter containing zinc phosphide. Subsequent hydrolysis and absorption of phosphine may occur.4

Distribution
• Clinical signs in human poisonings suggest that phosphine is widely distributed to the liver, kidneys, and central nervous system.4

Metabolism
• Metabolism of zinc phosphide is not well understood.14

Excretion
• Phosphine may be exhaled from the lungs as the parent compound.4
• The primary metabolite excreted in animal urine is hypophosphite.4

Medical Tests and Monitoring:
• Tissue samples taken from suicide victims were analyzed using headspace gas chromatography with inductively coupled plasma mass spectrometry or a nitrogen-phosphorus detector following the fatal ingestion of aluminum phosphide tablets. Phosphine residues were detected in the brain, kidney, liver, heart, and surrenals. Phosphorus and aluminum residues were detected in blood.40,41

• Silver nitrate strips have also been used to detect phosphine in post-mortem tissue samples. However, the process of anaerobic decomposition may produce traces of phosphine that are independent of any exogenous exposure.42
• These biomarkers have not been widely utilized.

Environmental Fate:

Soil
• Zinc phosphide oxidized in 5 weeks when placed in soils that had at least 50% moisture saturation.4
• Three volcanic soil types with organic matter ranging from 3-15% were mixed with water to 25, 50, 75, and 100% saturation. Researchers then added 4.17 mg/g zinc phosphide to the soil and sealed the mixtures in glass vials. The maximum amount of phosphine liberated from one saturated soil type was 32% of the total amount possible based on phosphorus content of the zinc phosphide. Phosphine production from the soils peaked at 1-9 days after addition, depending on soil type and moisture content.43
• In the same study, researchers added zinc phosphide to dry soils and sealed the mixture in glass vials. During the incubation process, no phosphine was detected in the headspace of the vials. The researchers concluded that zinc phosphide may have oxidized to zinc phosphate, and that any phosphine produced during the decomposition subsequently oxidized as well.43
• Phosphine diffuses into the voids within soil but this process is reduced with increasing soil moisture levels.43 Phosphine absorbed by soils is subsequently oxidized to orthophosphate.4
• Five commercial zinc phosphide baits placed on silt clay loam from harvested sugar cane fields retained 50-100% of the original zinc phosphide content at the end of 16 days. The loss was greatest in the oat bait that had a surface treatment of zinc phosphide.44
Water

- Zinc phosphide placed in fresh or salt water for 11 days showed little hydrolysis.4

- Hydrolysis of zinc phosphide is substantial only in highly acidic conditions of pH 4 or below.4

- Sediments in water are expected to facilitate the breakdown of zinc phosphide to either phosphine under anaerobic conditions, or phosphoric acid under aerobic conditions.4

- Phosphine will oxidize in water to form hypophosphorus acid.4

Air

- Phospine released into the air rapidly breaks down following reaction with hydroxyl radicals, with a half-life of 5-28 hours. The oxidation products are inorganic phosphate and phosphorus oxyacids.4 See the text box on Half-life.

- Phosphine will spontaneously combust at concentrations of greater than 1.8% and temperatures of 38 °C (104 °F). If other phosphorus hydride impurities are present, phosphine may ignite at room temperature.5

Plants

- Commercial rodenticide pellets containing 2% zinc phosphide were placed in artificial gopher burrows in an alfalfa field. Pellets were applied at 3 pounds per acre (the label rate), 6 pounds per acre, and 9 pounds per acre in burrows 8” deep and 10’ apart. Above-ground portions of the alfalfa were harvested after 1, 2, 7, and 30 days. No residues of zinc phosphide were detected in the alfalfa.45

- A rodenticide bait containing 2% zinc phosphide was broadcast at rates of 5, 10, and 50 lbs/acre on sugarcane in Hawaii four times at two-month intervals. Samples of vegetation were taken one week after the final application and at harvest, 110 days later. The samples at one week contained 0.004-0.015 ppm of phosphine at the dry site and 0-0.045 ppm of phosphine at the wet site. At harvest, phosphine residues at the dry site were 0.006-0.032 ppm. No residues were detected at the wet site at harvest.46

Indoor

- No data were found on indoor fate of either zinc phosphide or phosphine.

Food Residue

- The USDA does not monitor food samples for zinc phosphide or phosphine as part of the Pesticide Data Program.47,48 Phosphine tolerances from the use of zinc phosphide are established for hay, wheat, alfalfa, barley, potatoes, grapes, sugar beets, and beans.49

- The U.S. EPA determined that neither acute nor chronic exposure to zinc phosphide was expected through the diet.2

Ecotoxicity Studies:

Birds

- The acute LD₅₀ for northern bobwhite quail (Colinus virginianus) is 12.9 mg/kg50, and the LD₅₀ for mallard ducks (Anas platyrhynchos) is 67.4 mg/kg.51 The 5-day LC₅₀ was 469 ppm in bobwhite quail and 2885 ppm in mallards.52,53

- The LD₅₀ values for other bird species ranged from 7.5 to 12.0 mg/kg for three species of geese (Canada goose Branta canadensis, white-fronted goose Anser albifrons, and snow goose A. caerulescens) and 24 to 178 mg/kg for red-winged blackbirds (Agelaius phoeniceus).19,54
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- Canada geese (Branta canadensis moffitti) and white-fronted geese (Anser albifrons) were placed in pens in alfalfa and fescue fields that were treated with a 1% zinc phosphide grain bait. The exposure interval was 4 days. Four of the six Canada geese died following exposure to grain bait on fescue, and the rest lost weight. White-fronted geese exposed to bait on alfalfa all survived, although the geese did consume sublethal amounts of the bait. The white-fronted geese appeared to develop an aversion to the bait, which may have been due to the strong emetic action of zinc phosphide. The researchers concluded that insufficient forage leads to greater likelihood of bait ingestion.54

- Wild geese and domestic fowl exposed to repeated sublethal doses of zinc phosphide appeared in some cases to develop diarrhea. Researchers noted increased excretion of bile pigments in the droppings of the domestic fowl.54,55

- Researchers fed great horned owls (Bubo virginianus) for 3 days exclusively on voles killed with 87 mg/kg zinc phosphide 5 hours prior to being offered to the owls. The owls began roosting on the floor of their pens rather than on perches and refused to take flight when disturbed.33

Fish and Aquatic Life
- Phosphine is poorly soluble in water, but when it is in solution it can be acutely toxic to aquatic life.4
- The 96-hour LC₅₀ for phosphine exposure in rainbow trout (Onchorhynchus mykiss) was 0.0097 ppm. The EC₅₀ for Daphnia exposed to phosphine in a 24-hour test was 0.2 mg/L.1 See the text box on EC₅₀.
- An acute LC₅₀ for rainbow trout exposed to phosphine was reported as 0.5 mg/L, and for bluegill sunfish (Lepomis macrochirus) the LC₅₀ was 0.8 mg/L.1

Terrestrial Invertebrates
- No information was found on the effects of zinc phosphide or phosphine on terrestrial invertebrates.

Regulatory Guidelines:
- The chronic reference dose or cRfD for zinc phosphide is 0.0001 mg/kg/day.2 See the text box on Reference Dose (RfD).
- The U.S. EPA did not classify zinc phosphide or phosphine with regards to carcinogenicity because chronic exposure is not expected.2 See the text box on Cancer (page 5).
- The National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Limit, Time-Weighted Average (REL, TWA) for phosphine is 0.4 mg/m³.51
- The Permissible Exposure Limit (PEL) for phosphine is 0.4 mg/m³.56
- The Acute Exposure Guideline Levels, or AEGLs, for zinc phosphide range from 3.6 ppm for AEGL-3 for a 10-minute exposure to 0.13 for an 8-hour exposure, AEGL-2.57
- The ACGIH Threshold Limit Value, or TLV, for phosphine is 0.3 ppm or 0.4 mg/m³.58
- The exposure level considered Immediately Dangerous to Life and Health, or IDLH, for phosphine is 50 ppm.58

Date Reviewed: September 2010

References


47. **Pesticide Data Program Annual Summary, Calendar Year 2006; U.S. Department of Agriculture, Agricultural Marketing Service: Washington, DC, 2007.**


